new form of irregularity often presents itself in coupled beats or continuous bigeminus in which each beat of the ventricle that arises in the normal way from an auricular impulse is followed by a contraction arising from the ventricle itself.

In connection with the treatment of fibrillation with digitalis, I may again remind you that an attack of this condition may in certain circumstances be precipitated by this drug.

In animal experiments various other measures have been found useful in arresting fibrillation, but these are all inapplicable in human patients. Thus I have found that cooling the heart with iced saline is often successful, while Gottlieb found that camphor perfused through the vessels seemed to have some effect in retarding its development. Hering recommends the perfusion of potassium chloride until the heart ceases all movement, and then restores it by means of Ringer's solution perfused through the coronaries. I merely mention these methods to show their inapplicability, so that no one may be induced to attempt the treatment of this condition by means of potassium salts.

PERFORATED ULCER OF THE STOMACH AND DUODENUM.

By George G. Ross, M.D.,
SURGEON TO THE GERMANTOWN HOSPITAL; ASSISTANT SURGEON TO THE GERMAN HOSPITAL, PHILADELPHIA.

Within the past two years I have had four cases of perforating ulcer of the stomach or duodenum, several of which show points of marked interest in reference to the diagnosis, treatment, and end results. They are as follows:

Case I.—Mr. Samuel M., referred by Drs. Ullom and Johnson, was admitted to the Germantown Hospital September 17, 1908. He gave a history of chronic dyspepsia, associated with vomiting and localized pain in epigastrium. He had been a sufferer from these symptoms for years. At 11 A.M. on the morning of admission he was seized with sudden, agonizing pain in the upper abdomen. The pain, which was extremely severe, radiated to the back and seemed to have its point of greatest intensity in the right iliac fossa. His abdomen was generally rigid, being most marked in the epigastric region. He was exquisitely tender over McBurney's point. There was no very great tenderness of the upper abdomen. He was operated on at 1 P.M., two hours after the onset of the attack. When the peritoneum was opened, gas and gastric contents escaped. The greatest quantity of fluid and debris was found in the right iliac fossa, although there were large quantities throughout the peritoneal cavity. A hole large enough to thrust one's thumb through was discovered in the duodenum, one-half inch from the pylorus. This was closed by through-and-through sutures and oversewn with two layers of Lembert's suture. The drainage was placed through the wound to the site of the perforation and there was drainage through a button-hole incision above the pubes. For the first forty-eight hours after operation the patient had anuria, after which time the kidneys began to functionate. There was no evidence of either leakage or peritonitis. The material from the drains consisted of serum, with a small amount of stomach contents. Gastro-enterostomy was not done on this patient for the reason that the anesthetist reported the man as dead when the perforations had been closed. I believe, in view of the subsequent events, that had it been possible to have performed a gastro-enterostomy, the man might have recovered.

Seven days after the primary perforation the patient had a second perforation, with the same characteristic symptoms, resulting in death. The postmortem examination showed that there had been no leakage through the original perforation, and, while there was very little healing, the stitches had held it tight. The second perforation had occurred three-fourths of an inch from the first one, and when the duodenum was opened it was discovered that both perforations had occurred in the same ulcer, which was horseshoe-shaped, corresponding to the curve of the duodenum. There were four other ulcers of the duodenum. Dr. Ullom examined the specimen under microscope and discovered no malignant change.

CASE II.—Virgil H., aged forty years, was admitted to the German Hospital November 11, 1908; discharged January 16, 1909. The history was unfortunately overlooked and can be remembered only in outline. The family history is unimportant. For some years the patient had been troubled with indigestion, distress after eating, and pain in the epigastrium, with eructations of gas and occasionally nausea and vomiting. He had been treated palliatively and had tried sanatorium treatment at Battle Creek and the use of health foods without success. Ten days before admission the patient, while walking along the street, was seized with severe pain in the hypochondrium, radiating to the back and the right chest, accompanied with nausea and vomiting, and since then he has had chills, fever, and sweats. Bowels regular. Deep breathing gave him pain in the right hypochondrium. He gradually became greatly prostrated and weak. There was a tender localized mass in the region of the gall-bladder and a tentative diagnosis of empyema of the gall-bladder was made.

On November 12, under ether anesthesia, an incision was made through the upper right rectus. The pylorus and duodenum were found plastered tightly to the under surface of the liver, which was itself quite adherent to the abdominal wall. Upon gently releasing these adhesions, a large subphrenic abscess was encountered lying beneath the right vault of the abdomen, and a considerable quantity of yellowish, foul-smelling pus was evacuated. There was some soiling of the general peritoneal cavity. On the anterior surface of the stomach, about three inches from the pylorus, a small perforation was found, which had been closed by the adhesions which bound the stomach to the edge of the liver. The edge of the perforated ulcer was turned in by chromic gut suture and oversewn by Lembert sutures of linen thread. The abscess cavity was drained by a rubber tube, emerging through the anterior wound, and a tube was also inserted through a counter puncture through the right loin. Three pieces of gauze were placed about the stomach, pylorus, and subhepatic surface. Fearing soiling of the lower abdomen, a glass tube was inserted in the pelvis through a suprapubic stab. The incision was partly closed by through-and-through sutures of silkworm gut.

The patient recovered without any unusual symptoms, and upon leaving the hospital had a small discharging sinus above the pubis.

Five months after the date of operation a piece of gauze one inch wide by five inches long was passed through the suprapubic sinus, which had persisted since the removal of the glass drainage tube. It was a piece of gauze of the tube dressing which had been cut thin at one place and had become separated and at the next dressing had been pushed through the tube and left there when the tube was withdrawn. Since this time all tubes at the German Hospital are dressed with selvage gauze.

On the day of admission the leukocyte count was 25,280 and the hemoglobin 58 per cent. The urine showed a slight trace of albumin and casts for three weeks after the operation. A culture from the pus at the time of operation showed staphylococcus.

CASE III.—Andrew B., aged fifty-five years, was admitted to the Germantown Hospital January 16, 1909; discharged January 23, 1909. The family and early personal history is of no importance. For six months prior to admission the patient had been suffering from dyspepsia. Two years ago he had a serious and severe accident, sustaining fractures of the ribs, clavicle, and right leg. At half after four o'clock on January 15, 1909, while in a barber chair, the patient was seized by a sudden, sharp, severe pain in the upper right abdomen. He was able to walk to his home about one block away. Family remedies failing to give relief, he sent for Dr. Funk about 7.30 P.M. At this time his greatest pain and tenderness had localized in the right iliac fossa. On admission he had marked tenderness and rigidity in the right iliac fossa. Temperature, 100.2°; pulse, 100, and respiration 32.

He was operated on at once, an incision being made through the right rectus muscle, as we believed we had an appendiceal abscess to deal with. As soon as the peritoneum was incised, gas and pus escaped from the cavity. A congested appendix containing blood-clots was removed. The incision was enlarged upward and plastic exudate was found under the liver and about the stomach. A perforated ulcer was found on the anterior surface of the lesser curvature of the stomach which was closed by chromic gut suture, and over-whipped by Lembert suture of silk. Cigarette and gauze drainage was inserted at the upper end of the incision and a glass drainage tube was inserted into the pelvic cavity. The wound was sutured between the drains. The patient apparently held his own until November 22, 1909, and then failed rapidly and died November 23, 1909, at 6.30 P.M.

Postmortem Examination. The ulcer in the stomach which had been sutured was found healed. A perforated ulcer was found in the duodenum about 2 cm. from the pylorus. There was considerable pus in the peritoneal cavity, and the intestines were matted together by plastic exudate. An indurated and suppurating area was found in the lower lobe of the right lung.

This case illustrates several important aspects of peptic ulcer: (1) The combined peptic ulcer of the stomach and duodenum; and (2) a primary perforation of a gastric ulcer and a subsequent perforation of a duodenal ulcer. Here again the value of a gastro-enterostomy presents itself. Temporary drainage by gastro-enterostomy might have prevented the second perforation.

CASE IV.—Joseph A., aged thirty-four years, born in Russia, was admitted to the German Hospital as a case of acute appendicitis with beginning peritonitis. The patient had felt well, aside from a poor appetite, until 2.30 P.M. on the day of admission. He had a piece of meat and tea for breakfast; nothing for dinner. At 2.30 P.M. he drank a glass of soda water and almost immediately was stricken with extremely severe pain across the right upper abdomen, which radiated into the right side of the back. There was some general abdominal pain but it was most intense in the upper abdomen. He was weak and perspiring profusely. He was doubled up like a jackknife, but obtained no relief. The bowels moved well in the middle of the forenoon. No definite gastric history could be obtained. Two weeks ago the patient began to belch up a little gas, and his appetite was poor. He complained of an occasional vague pain in the epigastric region. The only noteworthy thing about this pain was that on several different occasions it was relieved by vomiting. The patient never noticed blood in the vomitus or stool. No history of gall-bladder or appendiceal symptoms.

On admission, at 5.30 P.M., the patient had an anguished countenance, was restless, and complained of paroxysmal abdominal pain of severe character. Features were thin; his respiration was rapid and thoracic in type. Abdomen scaphoid, with board-like rigidity in the upper half; less marked but present in the lower half. Extreme tenderness above umbilicus, less marked below this point.

No more tenderness existed over appendix than in corresponding point on the left side. Liver dulness extended from the sixth rib to costal margin. The flanks were clear and soft, but a tender tympanitic swelling corresponding to the size and shape of the inguinal canal was found in that area on the right side. This felt like fluid under pressure and was easily reduced, but returned immediately on the release of pressure.

An operation was performed, with the diagnosis of perforated peptic ulcer. The patient took ether badly and in large quantities. On making an incision through the right rectus, some yellow, cloudy fluid was seen, and the bowel was slightly congested and distended. The appendix was examined and found negative, but it was removed in the usual fashion, but with some difficulty, on account of the high incision. The stomach examination was negative. The duodenum was examined and a large indurated ulcer was found in the anterior surface of the duodenum about 1 cm. from the pylorus. The ulcer was about 3 cm. in diameter. In the centre of it was a small pinhead perforation through which was escaping gas and fluid. A large gray area about 1.5 cm. in diameter, surrounding the perforation, was just about ready to slough out. The whole area was turned in with silk sutures, but this was difficult, on account of the poor quality of the tissue. A posterior gastro-enterostomy was done in the usual manner. A glass tube was inserted into the pelvis through a suprapubic incision and about 50 c.c. of cloudy yellow fluid was withdrawn. The tube was removed. The neighborhood of the perforation was mopped gently and the upper incision closed by tier sutures. The Fowler position and the Murphy treatment were employed after the operation. The further course was uneventful.

On admission the temperature was 99°, the pulse 84, and the respiration 26. A culture from the free abdominal fluid, made on November 22, was sterile. Examination of the appendix showed chronic interstitial appendicitis.

There is not any condition which the abdominal surgeon is called upon to treat in which prompt diagnosis, properly instituted treatment, and correct operative technique are more essential than in perforated ulcer of the stomach and duodenum. Ulcer of the stomach and of the duodenum can be considered together, not only because their pathology is practically identical, but also because, in view of the close relations of the two organs and their practically identical relations to other abdominal organs, the symptoms of perforation and the treatment thereof are practically the same.

THE FREQUENCY OF PERFORATION. The frequency of perforation of either gastric or duodenal ulcer is almost impossible to determine with any degree of accuracy. It has been estimated[1] that 7 per cent. of gastric ulcers will perforate. It is very doubtful if this could be verified. Clinically, the number of perforations in a series of cases of gastric ulcer not previously treated surgically, will, I believe, be found to be considerably higher.

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[1] Pariser and Linder. Quoted by Deaver and Ashhurst.

Upon duodenal ulcer there are practically no reliable statistics as to the frequency of perforation. It should also be borne in mind that up to the last few years many ulcers which were doubtless duodenal had been classed among "gastric ulcers of the pyloric region." Thus, some years ago duodenal ulcer as compared to ulcer of the stomach was considered rare; whereas now many operators, such as Mayo,[2] Moynihan,[3] and Mayo Robson, whose experience in surgery of the upper abdomen is most extensive, now regard it as being more frequent than gastric ulcer. William Mayo has called attention to a possible explanation of this. In the Mütter Lecture before the College of Physicians of Philadelphia in 1908, he drew attention to the fact that the dividing line between the stomach proper and the duodenum is indicated by a sharply defined but not generally recognized line, recognizable superficially by the course of the pyloric veins. Previously the distinction between the duodenum and the stomach had been upon general lines, and in doubtful cases the diagnosis of gastric ulcer had been preferred to that of duodenal ulcer.

It must be stated, however, that in many series of perforated ulcers of the stomach and duodenum the number of perforated gastric ulcers is so far in excess of the ulcers of the duodenum that it seems doubtful whether duodenal ulcer is really so frequent as is stated by Mayo and Mayo Robson. Thus, in the experience of Miles,[4] who published the largest series of perforated ulcers of the stomach and duodenum which have occurred in any one individual practice, of 46 perforated ulcers but 10 were duodenal, while 36 were gastric. In a series of 13 cases of perforated peptic ulcer operated upon at the German Hospital since 1904, 10 were duodenal and 3 gastric.

LOCATION OF THE PERFORATION. The location of the perforation, both in the stomach and duodenal ulcer, is almost invariably on the anterior surface of the organ affected. Perhaps this is because the anterior surface lacks the same support that is given by the underlying structures to the posterior walls. Thus, in statistics collected by Ashhurst and Deaver,[5] 60.5 per cent. of duodenal ulcer perforations were upon the anterior wall, while the same was true of 71+ per cent. of gastric ulcers. By far the greatest number of duodenal ulcers were in the first part of the duodenum.

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[2] Jour. Amer. Med. Assoc., 1908, p. 556.

[3] Medical Press and Circular, London, 1908, n. s., lxxxvi, 110.

[4] Edinburgh Med. Jour. 1908, n. s., xx, 106–117.

[5] Surgery of the Upper Abdomen, 1909.

SYMPTOMS OF PERFORATION OF GASTRIC AND DUODENAL ULCERS. The symptoms of the perforation of an ulcer of the stomach or duodenum vary. A perforation may be either acute or rapid, chronic or slow. Rapid perforations are particularly those in which the rupture of the viscus takes place into the free abdominal cavity so suddenly that no restraining barriers of adhesions have been previously formed. Such were Cases I, II, and IV of the series which I report.

The symptoms of a rapid gastric or duodenal perforation are very similar. The first indication of perforation may come at the time of the actual perforation, although in a certain number of instances premonitory signs, such as epigastric fulness and distress, make known the fact that there is some unusual disturbance of the diseased area.

Whether or not these premonitory signs are present, the first *important symptom is a sudden sharp and agonizing pain referred* to the epigastrium. The patient is invariably prostrated by the pain. Moynihan mentions several instances in which patients apparently succumbed to the shock caused by the extremely severe initial pain. The pain is, as a rule, well localized to the epigastrium, but may be referred, as in Case IV of the present series, where it was referred to the right dorsal region. After a comparatively short time the most acute pain leaves the patient, but he still has a marked feeling of pain, tenderness, and heaviness in the epigastrium. Vomiting in duodenal ulcers is so uncertain that it is not a symptom of the first importance. Hematemesis is rare in duodenal ulcers but comparatively common in gastric perforation. Elder, however, has reported a case of acute duodenal perforation in which the patient vomited bright red blood. This symptom is deceptive and hardly worthy of very great attention.

It was formerly stated that almost invariably there followed immediately with or after the pain a condition of shock. It is doubtless true that we find extreme prostration and weakness of the patient. The face is drawn and anxious, the patient perspires freely, and is somewhat pale or even slightly cyanosed in appearance. But as regards the pulse, temperature, and respiration, there is no evidence of shock until later, when a beginning septic peritonitis may give those symptoms. Thus, in Case IV, we find in an acute duodenal perforation, seen early, a temperature of 99° F., a pulse of 84, and respirations of 26 to the minute—certainly not significant of what is ordinarily considered as shock. The subjective symptoms, however, are not more important than those which we can discover upon thorough examination of the patient.

Abdominal palpation reveals an abdomen in which there is board-like rigidity of the recti, especially the right, and most marked in the upper abdomen. This extreme rigidity, which I have noted in my cases, is the most important diagnostic feature. The rigidity accompanying perforated ulcer in the upper abdomen far exceeds that of anything but a most unusual appendicitis or gall-bladder lesion. In association with the rigidity we find early an abdomen which may be somewhat scaphoid, but as soon as the peritoneal reaction sets in distention begins. The abdominal muscles are held rigid to protect the underlying diseased area. The diaphragm is also in close relation to the infected field, therefore we find, as we would expect, that the patient's respirations are shallow and thoracic. This is due to nature's effort to put a muscular splint upon the whole peritoneal area surrounding the acute lesion. With the marked rigidity of the upper abdomen we note a general abdominal rigidity also—and one which gradually increases as the products of peritoneal infection spread over the whole abdomen.

Tenderness in the epigastrium in cases of perforation is to be marked early and is most severe. It has been stated that in duodenal perforation the tenderness is somewhat more to the right of the median line, while in perforations of the stomach it is to the right if the pyloric or prepyloric area be affected, and to the left if the lesion be in the body or either one of the curvatures of the viscus. The area of greatest tenderness, however, in either gastric or duodenal perforation may be about the umbilicus, and has even been referred to either iliac fossa, particularly, at times, to the right one. Leukocytosis is not always present immediately after the perforation, but appears as soon as peritonitis is in full activity. As it is an accompaniment of so many acute intra-abdominal inflammatory conditions, its usefulness is negligible and its only true value is as an indication of the patient's resistance. Moynihan and some others lay the very greatest stress upon anamnesis in the diagnosis of acute duodenal ulcer, and state that when a history of previous duodenal ulcer has not been obtained in those cases in which a perforated ulcer has been found, it is because the history has not been taken with sufficient care. In all my cases there was at least some history of gastric or digestive disturbance. Yet Elder,[6] Watson and Korte,[7] and many others mention instances in which the previous history does not point to ulcer of any sort. Korte, in a report of 18 gastric perforated ulcers and 1 duodenal perforated ulcer, says that of the 19 cases, 2 gave a history of very slight previous trouble, while 4 gave absolutely no history of disease in the upper digestive tract. We must make our diagnosis in acute perforation, then, largely upon the history of the immediate condition which causes the patient to seek treatment.

Case III of my series represents the class of subacute or slow perforations. These, as may be expected, are more common posteriorly where the adjacent viscera furnish support to the stomach and duodenum, and act as limiting structures to prevent the spread of infectious materials. In Case III, however, the ulcer was anterior. When a gastric or duodenal ulcer perforates subacutely, the symptoms are, as a rule, more protracted and less severe than in acute perforation. The course of the lesion subsequently is entirely different, for instead of the onset as a general or diffuse peritonitis, we have the formation of a localized abscess, sometimes anterior but usually subphrenic. The latter term as used generally will include also abscesses which have no real nearness to the diaphragm but are merely intraperitoneal abscesses of the upper abdomen. Mayo Robson states that perforated gastric ulcer is the most frequent cause of subphrenic abscess—a statement that Deaver does not agree with, as in his experience appendicitis is the most common cause.

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[6] Annals of Surgery, 1906, xliii, 390.

[7] Archiv f. Chirurgie, vol. lxxxi; Berl. klin. Wchnschr., 1907, xliv, 226.

In addition to the general signs of perforation in such instances, we would have the local signs of a circumscribed collection, particularly if this be anterior. The whole course of the illness is less acute, and since the patient does not develop an acute and rapidly spreading peritonitis, we have the clinical picture of localized abscess, with systemic infection of the septic type. Chills, irregular fever, sweats, leukocytosis, etc.

DIFFERENTIAL DIAGNOSIS. The differential diagnosis of perforated gastric and duodenal ulcer is, as a rule, not difficult. There are, however, certain conditions which must be eliminated. Biliary colic may give pain almost as severe, and even temporary prostration as marked as that of perforated ulcer, but the subsequent hour or two with the failure of the case to show evidence of upper abdominal peritonitis will surely clear the diagnosis. Rupture of the gall-bladder is rare in the absence of definite history of previous enlargement and inflammation of the viscus. It is also absolutely far more rare than rupture of gastric or duodenal ulcer. Should it, however, occur in any instance in which the previous history did not point to a gall-bladder condition, I hardly see how it could be absolutely differentiated. In either case operation is indicated, so that the distinction is of no great importance. The conclusion that the primary pain of perforating ulcer cannot always be distinguished from that caused by disease of the biliary organs has been voiced also by Van Swerigen.[8]

The ileus may be considered to be the point of origin of the sudden severe pain. It should not, I believe, be difficult to differentiate even the most suddenly beginning intestinal obstruction from perforating ulcer. But it is interesting to note in this connection that in a series of 11 cases reported by Van Khantz,[9] 2 were supposed to have had their symptoms caused by obstructions at the site of an old hernia. Probably poor history taking and rapid and superficial examination account for this error.

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[8] Jour. Amer. Med. Assoc., Chicago, 1908, li, 405.

[9] Archiv f. klin. Chir., 1908, lxxxv, 700–717.

Menstruation is mentioned by Moynihan as having three times in his experience given symptoms which led to the diagnosis of perforated gastric ulcer. This, however, is a most unusual error in diagnosis. Perforation of malignant growth of the large and small intestines cannot, of course, be accurately diagnosed or distinguished from other abdominal perforations unless previous history and examination point definitely to their existence.

Perforated jejunal peptic ulcer is identical in pathology and anatomy, and therefore also in symptomatology and treatment, to perforated gastric and duodenal ulcer.

Acute appendicitis is the diagnosis most frequently made where, in cases of perforated gastric and duodenal ulcer, a correct diagnosis has not been made. One of the four cases of my series was so diagnosed before being sent into the hospital. That this is not unusual will be seen by the appended table:

Operator or reporter.	Cases of perforated ulcer.	Diagnosis of appendicitis.
Von Khnatz	11	3
Peck[10]	7	3
Federmann[11]	16 (op. 11)	2
Korte	19	2

The reason for this is twofold: (1) Acute appendicitis is the most common cause of intraperitoneal acute inflammation; and (2) perforated gastric and duodenal ulcer when discharging fluid or causing peritoneal infected fluid to form is, as a rule, so located that the fluids will gravitate toward the right iliac fossa.

Maynard Smith,[12] in a series of experiments carried out upon the cadaver, found that the fluid from such a source in a patient in a recumbent position followed the following course: (1) Downward in the direction of the right kidney pouch; thence (2) along the outer side of the ascending colon as far as the brim of the pelvis; thence (3) overflowed into the pelvis. Hence we can see why trouble of the appendix is so closely simulated, especially as Van Sweringen points out, after the lapse of about twelve hours.

It must not be forgotten that perforated duodenal ulcer and acute perforative appendicitis may be coincident. Indeed, Graham[13] reports a case in which a perforated retrocolic appendix formed part of the wall of an abscess originating from a perforated duodenal ulcer.

Acute pancreatitis has been mistaken for a perforated gastric or duodenal ulcer. There is no doubt that the early distinction is most difficult; but, as the case progresses, there is noted the gradual toxemia of pancreatitis, the absence of the characteristic rigidity of perforation, and the evidence of an infection of the omental bursa, and the marked symptoms of high intestinal obstruction—diaphragmatic pleurisy, diaphragmatic hernia.

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[10] Med. Record, N. Y., 1907, lxxii, 930–934.

[11] Deutsche Zeitschr f. Chir., 1907, lxxxvii, 443–481.

[12] Lancet, 1906, i, 895.

[13] Annals of Surgery, 1904, xl, 447.

Richardson[14] in a most careful article has drawn attention to the frequency with which acute intrathoracic disease simulates the symptoms of acute intra-abdominal lesions.

Barnard[15] reports an instance in which double basal pneumonia with a right diaphragmatic pleurisy in a patient with an unperforated gastric ulcer gave rise to symptoms leading to a diagnosis of perforation.

The appendix as a cause of disease simulating upper abdominal disease has come more and more into prominence within the last few years. I have already mentioned the frequency with which acute upper abdominal disease is diagnosticated appendicitis. While the reverse condition is not so common it must not be forgotten that appendicitis especially of the chronic variety often gives rise to crisis of pain referred to the upper abdomen occasionally accompanied by hemoptysis (Fenwick).[16] The occurrence of pylorospasm as a result of chronic appendicitis is not common but nevertheless is mentioned by several authors, amongst them Deaver and Ashhurst. It might even be possible that an acute fulminating attack of appendicitis might give rise to the symptoms of perforated gastric or duodenal ulcer, but we are far more likely to find it a confusing element in the anamnesis when attempting to elicit a history of upper abdominal disease prior to the suspected perforation.

Finally the possibility of a diaphragmatic hernia must be considered, although this condition is practically never diagnosed during life except by operation.

Hysteria or unperforated ulcer with peritonitis may all give rise to symptoms very closely simulating those of perforated ulcer.

The distinction between gastric and duodenal perforation is not always difficult; in fact, Moynihan considers it comparatively easy in most instances. Sometimes the anamnesis makes the case clear at once. At other times the distinction is more doubtful, because the history is less clear. In either case the practical importance of the matter is not very great because our treatment is the same for both gastric and duodenal ulcer.

TREATMENT. The treatment of perforated gastric and duodenal ulcer is in its main aspects entirely surgical. Nothing is surer than the fatal termination of a perforative peritonitis of the upper abdomen in all but the rarest instances when a surgeon is not put in charge of the case. The patient's only chance for recovery under medical treatment is in the formation of a localized abscess with spontaneous evacuation other than into the general peritoneal cavity, a termination so rare that it need not be considered.

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[14] Boston Med. and Surg. Jour., 1902, i, 399.

[15] Lancet, 1902, ii, 280.

[16] Dyspepsia, London, 1910, p. 78.

The time of election in operation for perforated gastric and duodenal ulcer is at once when the case is seen. All statistics agree that in those instances where acute perforation takes place and the infection is not circumscribed, early operations are by far the most successful. Thus, Van Khnatz had:

	Mortality, per cent.
5 cases operated in the first twelve hours; 3 died	60
1 case operated in the first eighteen hours; 0 died	0
5 cases operated after the first two days; 5 died	100

Or,

In the first twenty-four hours, 6 operated; 3 died	50
After forty-eight hours 5 operated; 5 died	100

Martens[17] had in 11 cases 6 recoveries, all operated upon from two and one-half to twenty-six hours after perforation.

Robson and Moynihan[18] give the following mortality in per cent. for perforated gastric ulcer: Under twelve hours, 28.5 per cent.; twelve to twenty-four hours, 63.6 per cent.; twenty-four to thirty-six hours, 87.5 per cent.; thirty-six to forty-eight hours, 100 per cent.; over forty-eight hours, 51.5 per cent had evidently had time to localize.

Patterson[19] in a consecutive series found in cases operated under twelve hours, 47 per cent. mortality; twenty-four hours, 50 per cent. mortality; twenty-four to forty-eight hours, 83 per cent. mortality.

There are a number of reasons why it is of the utmost importance to operate early in cases of gastric and duodenal ulcer. In the first place, it is well known that in any peritonitis beginning in the upper abdomen early operation offers the patient the very best chance of recovery. Early operation avoids the absorption of toxins and anticipates the loss of the patient's vitality caused thereby. It enables us to intervene when the disease process is still localized to some extent and has not reached the stage of general peritonitis. In cases of gastric and duodenal perforation it is even more important, however, to deal with the resultant peritonitis early than it is in inflammations of the lower abdominal or pelvic viscera. I have mentioned the path of diffusion of the toxic fluids formed or liberated within the upper right quadrant of the abdomen. There is in so many instances no attempt at localization that often we have a rapid spread of infectious material in a purely mechanical way. Naturally, if we operate in the first stage of a perforation, this mechanical diffusion will not be far advanced and we have to deal with a more or less localized peritonitis instead of one which is diffused or general.

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[17] Deutsche med. Wchnschr., 1907, xxxiii, 1851.

[18] Diseases of the Stomach, 1907, p. 101.

[19] Lancet, 1906, i, 575.

Moreover, it has been pointed out by Murphy[20] and others that the diaphragmatic zone of the peritoneum is by far the most absorptive area of the whole membrane. Infectious materials are absorbed with far greater rapidity by the diaphragmatic peritoneum than by any other portion, and not only is this true but we have found that the whole upper abdominal peritoneum shares to a certain extent this great power of absorption. Thus, while a pelvic or lower abdominal condition, such as appendicitis or salpingo-oöphoritis, occurs in the area in which there is a certain possibility of defense against toxins, exactly the reverse is true of a perforated gastric or duodenal ulcer. On this account, again, it is most important to remove both the source of infection and the infectious material already formed at the earliest possible moment. I have no doubt that when it becomes the usual thing among medical men and surgeons accurately to diagnose such conditions early and to operate at once under practically all conditions, the mortality will be reduced to a point far below that reached by any operator so far.

The operation itself must accomplish three things primarily: (1) It must include some form of closure of the ruptured ulcer; (2) it must adequately provide for the drainage, etc., necessary for the care of the peritoneal condition; and (3) under favorable circumstances, it may seek to prevent a recurrence of the perforative process. It is, of course, imperative that at the time of operation we should locate the perforation itself. After making the incision into the upper abdomen, a foul fluid, possibly containing portions of foodstuffs, may be discovered. If this be caused by a perforated ulcer of the alimentary tract, we often have also an escape of gas from the wound. Our diagnosis is then certain. Before proceeding any further we should at least attempt to prevent further diffusion of infectious materials during the operation itself by the proper use of large gauze pads to wall off the infected area. I am certain that the proper use of such barriers to infection in all operations for localized or even diffused peritoneal infections is of the utmost importance. It should be our object to introduce at once these pads, not only so that they effectually limit the spread of pus, etc., during the operation, but also that by their rough introduction and handling they do not denude the peritoneum of its endothelial coat and thus give rise to the subsequent formations of adhesions. If the rough handling of the clean peritoneum may give rise to the postoperative adhesions, these are certainly more apt to form when infection is already present. These pads also act as a sponge while in the general peritoneum.

When the operative area has thus been limited we may proceed in our endeavor to find and close the perforation. In cases beyond the first few hours there is often quite an amount of plastic lymph about the infected area. This should be removed only when it is in the road of the operator; otherwise it should not be disturbed, as there is reason to believe that it has a protective function. The omentum also has often come to the rescue and its adhesions should be gently separated so that we do not cause additional damage to the upper abdominal structures. Often, too, the omentum itself has covered the perforation and has acted as a temporary plug, preventing further escape of infectious material and localizing the peritonitis. By care in the use of our pads and careful though not slow operative procedures, it is often possible to complete the operation without further spreading the pus present.

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[20] Surg., Gynec., and Obstet., Chicago, 1908, vi, 575–598.

When the perforation has been found there are several points which must engage the surgeon's particular attention. In the first place, it is always wise to determine whether or not the perforation is single. It is true that multiple simultaneous perforation of the stomach or duodenum is the exception rather than the rule, yet they have been reported. Secondly, we should endeavor to make sure that the ulcer is not accompanied by others not yet perforated but about to do so. In two of my cases, both of them ending fatally, and the only ones so ending, death was caused by the perforation of a second ulcer. In Case II of perforated duodenal ulcer, a second perforation was found in the same viscus, while in Case III, in which the primary condition had been a perforated ulcer of the stomach, death resulted from the subsequent perforation of a duodenal ulcer. Ulcers evidently about to perforate should be treated as if they had already done so.

When we have found the seat of perforation, several methods of closure are open to us. It is my usual course to invaginate the ulcer as far as it is possible without occluding the lumen of the bowel and then to sew all layers with a continuous catgut suture, repeated if it is thought necessary to strengthen it. To make the closure tight, it is then completed by a continuos Lembert suture of fine linen thread. I do not believe it is essential to cut away the edges of the ulcer. If invagination is complete the sloughing edges will separate into the lumen of the bowel when healing takes place.

Eve[21] has lately suggested that the ulcerated area be excised by a longitudinal diamond-shaped incision, such as is used in pyloroplasty, and that the wound be then closed in the vertical direction, thus effectually avoiding narrowing the gut. In cases where such a narrowing seems especially likely, this method might be of use, but I have not as yet found it necessary.

When we have dealt with what appears to be the only perforation present we should investigate the viscus carefully to see if there is not another perforation. Gastric ulcers are multiple in about 8 per cent. of all cases; duodenal ulcers less frequently so; and perforations may also be multiple. Even when an ulcer has not yet perforated, it may be on the verge of doing so, and in that case we should deal with it exactly as if the perforation had already taken place. It must be remembered also that gastric and duodenal ulcers may exist coincidently, as in Case III, reported previously.

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[21] Lancet, London, 1908, i, 1822.

Adequate drainage is of the utmost importance. When the ulcers have been closed, provided that gastro-enterostomy has not been done, the peritoneal cavity may be cleaned in the immediate neighborhood of the field of operation by mild swabbing with gauze pads. I do not believe that irrigation of the whole peritoneal cavity is ever necessary or even advisable. At its best it cannot fail to disseminate infectious material, though not necessarily spreading infection. With proper drainage and modern postoperative methods, we are far safer in allowing the peritoneum to take care of itself. Irrigation may do good—it has often done harm—and the chances for the latter overbalance the possibilities of its beneficial action.

We are often best able to insert our permanent or postoperative drain before the gauze packs have been removed. Our drainage should do two things: Drain the original site of infection. This is best done by a rubber tube containing gauze, with plain gauze about, if necessary placed through, the incision itself. This may be replaced by a stab wound to one side of the incision, allowing the latter to be closed in its entirety and thus, perhaps, making the abdominal wall stronger. The choice must rest upon the judgment of the surgeon.

Körte[22] states that he considers a drain, as a rule, unnecessary but uses a pelvic drain where any drainage is indicated. I believe that we are usually safer in draining at the original site of infection. As the fluids of the upper abdomen always gravitate toward the pelvis, and particularly when we aid them in doing so by raising the patient in the bed, we should never force the pelvic peritoneum to deal unaided with the infectious material covering it. In all cases of severe, upper abdominal infection which might spread I employ a glass tube drain placed into the pelvis through a suprapubic stab wound and leave it in for two or three days until all danger of general peritonitis is past.

The question of gastro-enterostomy is one of great importance and is being largely discussed by surgeons today. Deaver and others claim that the drainage thus provided, be it temporary or permanent, will permit healing of the ulcer which may exist and thus prevent the liability to secondary perforation. Mayo and Moynihan do not advocate the procedure unless the lumen of the pylorus or duodenum is seriously diminished, as they claim that in the presence of a normally patulous duodenum or pylorus the artificial gastro-enteric opening will not functionate, and thus the object sought for will not be accomplished; and, as the operation takes time and must be done on a patient who is already hard hit, it to this extent lessens the patient's chance of recovery. This problem is like so many more in surgery in that it can and should be used in certain cases and is strongly contraindicated in others, and its successful use depends on the judgment of the surgeon.

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[22] Loc. cit.

The after-treatment of perforated ulcer consists in rest for the part involved by the avoidance of food ingestion and peristalsis and by the use of appropriate measures of defence for the peritoneum.

The patient occupies the high Fowler position so that the infectious fluid may gravitate from the absorptive diaphragmatic area to the safer pelvic zone. No fluid is given by mouth for thirty-six hours and the patient is kept thereafter on a liquid diet and semifluid for eighteen days. The Murphy method of giving saline by continuous enteroclysis is employed routinely; otherwise the treatment is that of any section case for a grave lesion. Vomiting, and even nausea, if at all persistent, should be treated by lavage and repeated as often as it is necessary permanently to relieve vomiting.

THE CELLULAR ELEMENTS OF THE BLOOD IN TUBERCULOSIS.

By Barton Lisle Wright, M.D.,
SURGEON UNITED STATES NAVY,
AND
Roscoe W. King,
UNITED STATES NAVAL HOSPITAL, LAS ANIMAS, COLORADO.

Red Cells. The various authorities upon this subject seem to agree in that there is but little change, if any, from the normal in the red count or hemoglobin percentage in tuberculosis, excepting in advanced cases shortly before death when both are decreased, sometimes to a marked degree.

In the routine examination of the blood of cases admitted to Las Animas Hospital, we found this to be so uniformly true that we discontinued making the red count and the estimation of hemoglobin as routine procedures. In a large number of cases the reds averaged above 5,000,000 per cubic centimeter and the hemoglobin 85 per cent.

It has been found, however, that the red cells are pathological to the extent of resisting the hemolytic action of salt solution at least, and from this fact a method[1] of obtaining the hemolytic index

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[1] The method for determining the hemolytic index is, briefly, as follows: Dilute the blood in the red cell pipette with an accurate 0.35 per cent. NaCl solution. Allow to stand five or ten minutes, and proceed to count the visible (non-hemolysed) red cells. Usually it will be possible, and preferable, to count the cells on that part of the slide used for counting leukocytes. In far advanced cases the central ruled area may have to be used. 150,000 divided by the number of cells found per cubic millimeter of undiluted blood will give the hemolytic index.